Revision 7

Store at -20C

#91068

TREM2 (D8I4C) Rabbit mAb

Cell Signaling TECHNOLOGY®

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For Research Use Only. Not for Use in Diagnostic Procedures.

Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W, W-S, IP, IF-IC, FC-FP	H	Endogenous	28	Rabbit IgG	#Q9NZC2	54209

Product Usage Information

Application	Dilution
Western Blotting	1:1000
Simple Western™	1:50 - 1:250
Immunoprecipitation	1:50
Immunofluorescence (Immunocytochemistry)	1:200 - 1:800
Flow Cytometry (Fixed/Permeabilized)	1:400

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.

Specificity/Sensitivity

TREM2 (D8I4C) Rabbit mAb recognizes endogenous levels of total TREM2 protein.

Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Leu221 of human TREM2 protein.

Background

The triggering receptor expressed on myeloid cells 2 (TREM2) protein is an innate immune receptor that is expressed on the cell surface of microglia, macrophages, osteoclasts, and immature dendritic cells (1). The TREM2 receptor is a single-pass type I membrane glycoprotein that consists of an extracellular immunoglobulin-like domain, a transmembrane domain, and a cytoplasmic tail. TREM2 interacts with the tyrosine kinase-binding protein DAP12 to form a receptor-signaling complex (2). The TREM2 protein plays a role in innate immunity and a rare functional variant (R47H) of TREM2 is associated with the late-onset risk of Alzheimer's disease (1,3). Research studies using mouse models of Alzheimer's disease indicate that deficiency and haploinsufficiency of TREM2 can lead to increased β-amyloid (Aβ) accumulation as a result of dysfunctional microglial response (4). These results agree with the distribution of TREM2 in human brain regions (e.g., white matter, the hippocampus, and neocortex) that are involved in Alzheimer's disease pathology (2). In addition, amyloid plaque formation induces expression of TREM2 and amyloid phagocytosis (5). Loss-of-function mutations in the corresponding *TREM2* or *DAP12* genes can result in Nasu-Hakola disease, a rare form of progressive presenile dementia that results from polycystic osseous lesions (6). TREM2 membrane shedding occurs by cleavage at the extracellular site between H157/S158, generating an N-terminal shedded fragment and a membrane bound C-terminal fragment (7,8).

Background References

1. Colonna, M. (2003) *Nat Rev Immunol* 3, 445-53.
2. Jonsson, T. et al. (2013) *N Engl J Med* 368, 107-16.
3. Boutajangout, A. and Wisniewski, T. (2013) *Int J Cell Biol* 2013, 576383.
4. Wang, Y. et al. (2015) *Cell* 160, 1061-71.
5. Melchior, B. et al. (2010) *ASN Neuro* 2, e00037.
6. Klünemann, H.H. et al. (2005) *Neurology* 64, 1502-7.
7. Thornton, P. et al. (2017) *EMBO Mol Med* 9, 1366-1378.
8. Schlepckow, K. et al. (2017) *EMBO Mol Med* 9, 1356-1365.

Species Reactivity

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

Western Blot Buffer

IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

Applications Key

W: Western Blotting **W-S:** Simple Western™ **IP:** Immunoprecipitation **IF-IC:** Immunofluorescence (Immunocytochemistry) **FC-FP:** Flow Cytometry (Fixed/Permeabilized)

Cross-Reactivity Key

H: Human

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